Article

Sudden acquired retinal degeneration syndrome in western Canada: 93 cases

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Abstract — This study reviewed clinical data from dogs diagnosed with sudden acquired retinal degeneration syndrome (SARDS) in western Canada. Medical records from the Western College of Veterinary Medicine from 2002 to 2016 showed that 93 cases of SARDS were diagnosed based on presentation for sudden blindness and a bilaterally extinguished electroretinogram. The most common pure breeds were the miniature schnauzer, dachshund, and pug. The mean age at diagnosis was 8.1 years and males and females were equally affected. Most of the dogs were presented with normal non-chromatic, but abnormal chromatic pupillary light reflexes. The incidence of retinal degeneration as detected *via* ophthalmoscopy increased over time after SARDS diagnosis. Polyuria, polydipsia, polyphagia, weight gain, elevated liver enzyme values, isosthenuria, and proteinuria were common clinical and laboratory findings. Chromatic pupillary light reflex testing may be more valuable than non-chromatic pupillary light testing in detecting pupil response abnormalities in dogs with SARDS, although electroretinography remains the definitive diagnostic test.

Résumé — Syndrome de la rétine silencieuse dans l'Ouest canadien : 93 cas. Cette étude a examiné les données cliniques provenant de chiens diagnostiqués avec le syndrome de la rétine silencieuse (syndrome de cécité soudaine acquise) dans l'Ouest canadien. Les dossiers médicaux du Western College of Veterinary Medicine de 2002 à 2016 ont montré que 93 cas du syndrome de la rétine silencieuse ont été diagnostiqués en se basant sur la présentation pour une cécité soudaine et un électrorétinogramme bilatéral sans incandescence. Les races les plus communes étaient le Schnauzer miniature, le Dachshund et le Pug. L'âge moyen au diagnostic était de 8,1 ans et les mâles et les femelles étaient également affectés. La plupart des chiens présentaient des réflexes pupillaires normaux à la lumière non chromatique mais des réflexes anormaux à la lumière chromatique. L'incidence de la dégénération rétinienne détectée par l'ophtalmoscopie a augmenté au fil du temps après le diagnostic du syndrome de la rétine silencieuse. La polyurie, la polydipsie, la polyphagie, le gain de poids, des valeurs d'enzymes hépatiques élevées, l'isosthénurie et la protéinurie étaient des résultats cliniques et de laboratoire communs. Le réflexe à la lumière pupillaire chromatique peut être plus utile que le test de la lumière pupillaire non chromatique pour détecter les anomalies de la réponse pupillaire chez les chiens atteints du syndrome de la rétine silencieuse, quoique l'électrorétinographie demeure le test diagnostique définitif.

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Introduction

Sudden acquired retinal degeneration syndrome (SARDS) is a prevalent cause of sudden, irreversible blindness in dogs (1–4) associated with apoptosis of photoreceptors of the retina (5,6). The condition was first described in 1984 (7) but despite considerable investigative efforts over the past 3 decades, its etiology and pathogenesis remain elusive.

This syndrome is currently diagnosed based on a history of acute vision loss, a fundic examination that lacks sufficient abnormalities to correlate with the degree of vision loss, and an extinguished electroretinogram indicating a lack of photoreceptor function. Significant retinal degeneration manifests weeks to months following vision loss (1,2,7). Dogs diagnosed with SARDS are frequently overweight, middle-aged or older, small breed, mixed-breed, and predominantly female (2,3,7–9). Breed predilections for SARDS have been reported in miniature schnauzers, cocker spaniels, dachshunds, Maltese, and pugs (2,4).

Affected dogs often present with clinical signs of polyuria, polydipsia, and polyphagia (8–11). Laboratory abnormalities in dogs affected by SARDS frequently include lymphopenia,

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Table 1. Proportions of the most common breeds diagnosed with SARDS and proportions of those same breeds examined by the Ophthalmology Service during the study period.

	SARDS		Ophthalmology referrals	
Breed	% (*n*/total)	95% CI	% (*n*/total)	95% CI
Miniature schnauzer	12.9 (12/93)	7.1 to 21.8	3.6 (201/5628)	3.1 to 4.1
Dachshund	10.8 (10/93)	5.5 to 19.3	1.2 (66/5628)	0.9 to 1.4
Pug	9.7 (9/93)	4.8 to 18.0	3.6 (201/5628)	3.1 to 4.1
Cocker spaniel	5.4 (5/93)	2.0 to 12.6	3.7 (207/5628)	3.2 to 4.2
Shih tzu	4.3 (4/93)	1.2 to 11.2	6.5 (364/5628)	5.8 to 7.1
Maltese	3.2 (3/93)	0.8 to 10.0	0.4 (24/5628)	0.3 to 0.6
Jack Russell terrier	3.2 (3/93)	0.8 to 10.0	1.5 (87/5628)	1.2 to 1.9

neutrophilia, proteinuria, increased serum cholesterol, alkaline phosphatase, aspartate aminotransferase, and alanine aminotransferase (1,2,8,9,11); however, it is uncertain how these are related to the disease. This syndrome is rarely a cause for enucleation or euthanasia (3) and affected eyes, therefore, are seldom available for histologic examination. Secondary ocular complications have not been reported with SARDS and most dogs adapt well to blindness. Consequently, many affected dogs are not re-evaluated by a veterinarian and are lost to follow-up.

In 2015, members of the American College of Veterinary Ophthalmologists identified SARDS as the ophthalmic condition with both the greatest requirement for, and potential benefit to be gained from, further research (1). There have been no SARDS-related studies conducted in Canada; therefore, this retrospective case series provides a valuable baseline for further research in addition to improving our understanding of SARDS within the broader global context. The objective of this study was to describe the signalment, onset, and duration of blindness, ocular examination abnormalities, and clinicopathologic findings of dogs diagnosed with SARDS at the Western College of Veterinary Medicine (WCVM).

Materials and methods

Electronic and paper medical records were searched to identify dogs diagnosed with SARDS at the WCVM in Saskatoon, Saskatchewan from January, 2002 to November, 2016. All cases met the study's inclusion criteria of having a diagnosis of SARDS based on presentation for blindness, an ophthalmic examination performed by a diplomate of the American College of Veterinary Ophthalmologists, a fundic examination that was either normal or mildly altered, and a bilaterally extinguished electroretinogram (ERG). Exclusion criteria included a duration of blindness prior to ophthalmic examination exceeding 6 mo, ERG recordings revealing asymmetrical retinal function, and fundic examination revealing moderate to severe retinal degeneration that could explain vision loss.

Data collected for each case meeting the inclusion criteria included signalment, geographic origin of the dog, month and year of presentation, concurrent systemic disease, medications and vaccinations administered 6 mo before diagnosis, ophthalmic examination findings, nature and duration of vision loss, pupillary light reflexes including chromatic pupillary light reflexes when available, and laboratory findings when available. The proportion of animals that had data available for each parameter was determined. Confidence intervals (95% CI) for proportions of each parameter were generated with R (version 3.3.1) statistics package. Means and standard deviations were calculated for age and duration of blindness before presentation, using SPSS version 24 (IBM, Armonk, New York, USA). A Mann-Whitney U-test for nonparametric data was used to compare the median duration of blindness in dogs with and without fundic changes using SPSS version 24 (IBM, Armonk). Statistical significance was set at $P < 0.05$.

Results

Ninety-three dogs met the inclusion criteria. Purebred dogs comprised 63.4% (59/93; 95% CI: 52.7 to 73.0) of the study population and were more common than crossbred dogs at 36.6% (34/93; 95% CI: 27.0 to 47.2). The most common breeds represented were the miniature schnauzer (12.9%, 12/93; 95% CI: 7.1 to 21.8), dachshund (10.8%, 10/93; 95% CI: 5.5 to 19.3), pug (9.7%, 9/93; 95% CI: 4.8 to 18.0), cocker spaniel (5.4%, 5/93; 95% CI: 2.0 to 12.6), shih tzu (4.3%, 4/93; 95% CI: 1.2 to 11.2), Maltese (3.2%, 3/93; 95% CI: 0.8 to 10.0), and Jack Russell terrier (3.2%, 3/93; 95% CI: 0.8 to 10.0) (Table 1). Of the 93 dogs, 46.2% were spayed females (43/93; 95% CI: 36.0 to 57.0), 41.9% were castrated males (39/93; 95% CI: 32.0 to 52.6), 6.5% were intact males (6/93; 95% CI: 2.6 to 14.0), and 5.4% were intact females (5/93; 95% CI: 2.0 to 12.6). Age at the onset of blindness ranged from 3 to 16 y, with a mean age and standard deviation at diagnosis of 8.1 +/− 2.5 y. Dogs aged < 5 y, 6 to 10 y, and > 10 y comprised 10.8% (10/93; 95% CI: 5.5 to 19.3), 75.3% (70/93; 95% CI: 65.0 to 83.3), and 14.0% (13/93; 95% CI: 7.9 to 23.0), respectively. Most dogs originated from Saskatchewan (68.8%, 64/93; 95% CI: 58.2 to 77.8), followed by Manitoba (22.6%, 21/93; 95% CI: 13.9 to 31.4), Alberta (6.5%, 6/93; 95% CI: 2.6 to 14.0), and British Columbia (2.2%, 2/93; 95% CI: 0.3% to 8.3%). The mean number of SARDS cases diagnosed per year was 6.2 +/− 3.7, ranging from 1 to 14 cases per year. A relatively even number of cases was diagnosed in the fall and winter [(Oct to Mar) (51.6%, 48/93; 95% CI: 41.1 to 62.0)] compared to the spring and summer [(Apr to Sept) (48.4%, 45/91; 95% CI: 38.0 to 59.0)].

As described by owners, the onset of blindness in SARDS dogs was sudden in 93.7% of cases (59/63; 95% CI: 83.7 to 97.9), progressive in 4.8% (3/63; 95% CI: 1.2 to 14.1), and episodic in 1.6% (1/63; 95% CI: 0.08 to 9.6). Duration of blindness was measured from the time the owners reported noticing their dog first went blind, as recorded in the medical history,

until the time of SARDS diagnosis. The mean duration of blindness prior to SARDS diagnosis was 36 +/− 4.5 d (n = 78; 95% CI: 27.5 to 45.4). Pupillary light reflexes (PLRs) with a white light were normal in 69.9% of cases (65/93; 95% CI: 59.3 to 78.7), incomplete in 25.8% (24/93; 95% CI: 17.5 to 36.1), and absent in 4.3% (4/93; 95% CI: 1.4 to 11.2). Chromatic or colorimetric PLR data were available for 12.9% of cases (12/93). Of these, PLRs with a red light were normal in 8.3% of cases (1/12; 95% CI: 0.4 to 40.2), incomplete in 33.3% (4/12; 95% CI: 11.2 to 64.5), and absent in 58.3% (7/12; 95% CI: 28.5 to 83.5). Pupillary light reflexes with a blue light were normal in 91.7% of cases (11/12; 95% CI: 59.7 to 99.5), incomplete in 8.3% (1/12; 95% CI: 0.4 to 40.2), and absent in none. Excluding age-related findings, cataract (37.6%, 35/93; 95% CI: 28.0 to 48.3) was the most common finding where incipient cataract was predominant (34%, 32/93; 95% CI: 25.0 to 45.0). Other concurrent ocular findings included corneal abnormalities (25.8%, 24/93; 95% CI: 17.5 to 36.1), conjunctival hyperemia (18.3%, 17/93; 95% CI: 11.3 to 28.0), adnexal abnormalities (15.1%, 14/93; 95% CI: 8.7 to 24.3), vitreal abnormalities (14.0%, 13/93; 95% CI: 8.0 to 23.0), and keratoconjunctivitis sicca (4.3%, 4/93; 95% CI: 1.4 to 11.2). A normal fundic examination was recorded in 52.7% (49/93; 95% C: 42.1 to 63.0) of cases. Ophthalmoscopic abnormalities observed in the remainder of cases included mild tapetal hyperreflectivity and/or mild vascular attenuation (34.4%, 32/94; 95% CI: 25.0 to 45.0), altered tapetal reflectivity (14.0%, 13/93; 95% CI: 7.9 to 23.0), and pinpoint retinal hemorrhage (2.2%, 2/93; 95% CI: 0.3 to 8.2). The median duration of blindness from cases without fundus changes was 14 +/− 4.5 d (n = 41) and differed significantly from the median duration of blindness from cases with fundus changes [30 +/− 7.8 d (n = 37) (P = 0.009)].

Information regarding medications or vaccines administered 6 mo prior to SARDS diagnosis was available for 38 dogs (40.9%). Of these animals, 36.8% (14/38; 95% CI: 22.3 to 54.0) received no medications and 63.2% (24/38; 95% CI: 46.0 to 77.7) received medications. A total of 31.6% of dogs (12/38; 95% CI: 18.0 to 48.8) received topical and/or systemic antibiotics, 28.9% (11/38; 95% CI: 16.0 to 46.1) received topical and/or systemic anti-inflammatory medication, and 5.3% (2/38; 95% CI: 0.9 to 19.0) received medication for hyperadrenocorticism in the 6 mo before diagnosis of SARDS. Dogs that were vaccinated 6 mo prior to diagnosis comprised 15.8% (6/38; 95% CI: 6.6 to 31.9) of cases.

Information regarding the presence of polyuria, polydipsia, polyphagia, and weight gain was available for 29.0% (27/93) of cases. Of these animals, 66.7% (18/27; 95% CI: 46.0 to 82.7) were polyuric and polydipsic, 51.9% (14/27; 95% CI: 32.2 to 70.8) were polyphagic, and 48.1% (13/27; 95% CI: 29.5 to 67.6) gained weight. Data were available for the presence or absence of concurrent systemic disease in 40.0% (37/93) of cases. Of these, 24.3% (9/37; 95% CI: 12.3 to 41.5) had urinary tract disease, 18.9% (7/37; 95% CI: 8.5 to 35.7) had dermatologic disease, 13.5% (5/37; 95% CI: 5.1 to 29.5) had gastrointestinal disease, 10.8% (4/37; 95% CI: 3.5 to 26.3) had cardiovascular disease, 5.4% (2/37; 95% CI: 0.9 to 19.5) had orthopedic disease, and 2.7% (1/37; 95% CI: 0.1 to 15.8) had diabetes mellitus. Among 6 dogs evaluated by adrenocorticotropic hormone stimulation testing (n = 3) or low dose dexamethasone suppression testing (n = 3), 2 were diagnosed with hyperadrenocorticism (5.4%, 2/37; 95% CI: 0.9 to 19.5). The dogs that tested positive for hyperadrenocorticism were not retested following their initial diagnosis.

The most common biochemical abnormality was an increase in 1 or more liver enzymes [alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyltransferase (GGT), glutamate dehydrogenase (GLDH), sorbitol dehydrogenase (SDH)] (71.4%, 15/21; 95% CI: 47.6 to 87.8) and hypercholesterolemia (47.6%, 10/21; 95% CI: 26.4 to 69.6). Where data were available, dogs diagnosed with SARDS with an abnormal USG (< 1.020) comprised 41.2% (7/17; 95% CI: 19.4 to 66.5), whereas dogs with a normal USG comprised 58.8% (10/17; 95% CI: 33.5 to 80.6). Of these 17 dogs, 76.5% had proteinuria (13/17; 95% CI: 50.0 to 92.2) and 23.5% did not have proteinuria (4/17; 95% CI: 7.8 to 50.2). Urine culture results were negative for all 5 dogs for which the test was performed.

Discussion

The proportions of breeds affected by SARDS in this study were consistent with similar reports indicating a preponderance for mixed breeds and pure breeds including dachshunds, miniature schnauzers, pugs, cocker spaniels, and Maltese (2–4,9,12–14). When considering all pure breeds as a group, our results indicate that pure breed dogs are more commonly affected than are mixed breed dogs. The mean age at SARDS diagnosis in our study was 8.1 y, which is similar to other reports in which the mean age at the time of SARDS diagnosis was between 8 and 9 y (2–4,8,9,12–14). Females are often said to be overrepresented in dogs with SARDS (3,4,8,9); however, the proportion of females in our study was 51.6% which aligns with other reports in which no gender predilection was found (2,12). Most of the dogs we examined originated from Saskatchewan, and the provincial distribution of cases observed in the study is a result of the geographic location of the veterinary hospital and reflective of our general referral population. Similar to other studies (3), we observed an even distribution of SARDS cases over the course of the year which argues against seasonality as a factor in the incidence of SARDS, contrary to the original SARDS report in which clustering in December and January was observed (7).

The onset of blindness as reported by owners was sudden in most cases, which is an expected historical finding in SARDS. Seventy percent of dogs in our study had normal pupillary light reflexes, 25% had reduced pupillary light reflexes, and 5% had absent pupillary light reflexes in response to a non-chromatic, white light stimulus. Though this contradicts the generally accepted notion that the majority of SARDS dogs present with diminished pupillary light reflexes in response to white light (1,2,7), surprisingly little data are published on expected pupillary light reflexes in SARDS dogs. Differentiating rod-cone mediated pupillary light reflexes from intrinsic melanopsin-mediated pupillary light reflexes can be achieved by comparing pupil responses to red and blue light as red light has a wavelength that does not overlap with melanopsin sensitivity

(15). As such, pupillary light reflexes in healthy canine eyes are elicited with red and blue lights but in dogs with SARDS are only elicited by high-intensity blue light. This was shown in a study in which dogs with SARDS displayed miosis while dogs with optic pathway disease displayed mydriasis in response to a blue light stimulus (13). In our study, chromatic pupillary light reflex testing was initiated in the latter period of the study and thus performed on a small number of dogs but showed that only 8% had a normal pupillary light reflex with red light and over 90% had a normal pupillary light reflex with blue light. Our results, combined with previous studies, suggest that though colorimetric pupillary light reflexes are not completely reliable, they may be more useful than conventional white light-elicited pupillary light reflexes in the detection of pupil response abnormalities in dogs with SARDS. In addition, it is important to note that this specific chromatic pupillary light reflex finding is also observed in diseases such as retinal detachment in which the outer retina is affected and the inner retina remains intact (15). Thus, an extinguished electroretinogram in the face of a normal ophthalmoscopic examination is required to attain a definitive diagnosis of SARDS.

The most common concurrent ocular abnormality detected in our study was cataract at a rate affecting 38%, the majority of which were incipient in nature, which is comparable to other studies reporting concomitant cataract (2,8). Conjunctival hyperemia was found in 18% of dogs in our study, similar to a previous study which reported that 27% of dogs with SARDS displayed conjunctival hyperemia (2). Adnexal and corneal abnormalities were also reported in our study but, similar to other observed ocular comorbidities, were thought to be incidental findings. Fundic examinations revealed mild retinal degeneration manifested by mild tapetal hyperreflectivity and mild retinal vascular attenuation in 34% of cases, which is a similar proportion to some reports (2,9) but lower than other reports (7,8). The higher proportion of dogs with clinically observable retinal degeneration in these latter studies may be due to a longer duration of disease before examination. Indeed, the duration of blindness before diagnosis of SARDS was significantly longer in dogs with fundic changes (30 d) than in dogs with normal fundic examinationbs (14 d) in our study. This finding is in agreement with reports of ophthalmoscopic signs of retinal degeneration being visible only weeks to months after diagnosis of SARDS (1,7,15). Unlike ophthalmoscopic signs of retinal degeneration, pinpoint retinal hemorrhage observed in 2 cases were considered incidental findings. Despite a lack of follow-up, it is our impression that SARDS is a quiet ocular disease that does not result in secondary ocular complications (9).

Little data are available regarding exposure to medications, vaccines, or toxins prior to diagnosis of SARDS. The most common medications dogs with SARDS were administered in the 6 mo before diagnosis were topical or systemic antibiotics and topical or systemic anti-inflammatory agents. Sixteen percent of dogs were vaccinated in the 6 mo before diagnosis of SARDS. Conclusive evidence that there is a link between exposure to medicinal compounds and SARDS has yet to be presented.

Polyuria, polyphagia, polydipsia, and obesity are consistent findings in dogs with SARDS (2,3,7–9,11,16). In the cases for which data were available in our study, 67% were polyuric and polydipsic, half were polyphagic, and half had reported weight gain. Polyuria and polydipsia seem to be specific to dogs with SARDS as a previous study found polyuria and polydipsia to be reported in 38% of dogs with SARDS and only 5% of dogs with blindness due to neurologic disease (2). Conversely, obesity is a widespread clinical finding regardless of the presence or type of systemic disease and therefore is a nonspecific finding. Adipose tissue is an endocrine organ capable of both being influenced by glucocorticoids and causing hyperactivation of the hypothalamic-pituitary-adrenal axis and therefore multiple interactions may be at play (17–19). The literature often refers to hyperadrenocorticism-like clinical signs in dogs with SARDS and an association with hyperadrenocorticism (9). Interestingly, dogs with hyperadrenocorticism concurrent to noncompressive acute vision loss (SARDS-like) were reported to have elevated serum concentrations of cortisol, triglycerides, and glucose compared to dogs with hyperadrenocorticism without vision loss (10). The authors of this study proposed that these serum changes may have resulted in the observed altered retinal blood flow and may play a role in the pathogenesis of blindness in their study population. Despite these reports, hyperadrenocorticism is infrequently diagnosed in dogs with SARDS (1,3) and likewise, only 2 dogs in our study were diagnosed with and were receiving treatment for hyperadrenocorticism. It has also been suggested that a single adrenal function test in a case of SARDS is not definitive evidence of concurrent hyperadrenocorticism due to changes in the hypothalamic-pituitary-adrenal axis that may be a result of stress or other unrelated illness (11). Indeed, it is our impression that a positive test for hyperadrenocorticism warrants repeat testing to exclude a transient, stress-related response.

The most common systemic comorbidity identified in our study was urinary tract disease, followed by dermatologic disease and gastrointestinal disease. A plethora of systemic diseases has been reported to be present in conjunction with SARDS, including cardiovascular disease, hypersensitivity-related diseases, endocrinopathies, and periodontal disease (2,3,8) and are therefore thought to be incidental findings rather than playing a role in etiopathogenesis. The most common serum biochemical abnormality herein was an elevation of liver enzymes and hypercholesterolemia, which aligns with reports of patients with SARDS having a subclinical hepatopathy (2,7,8,14,16). An increased incidence of isosthenuria and proteinuria has also been reported similar to our study (2,8,9). However, age and breed matched control dogs were not represented in our study.

There is limited evidence to suggest that an immune-mediated process is at the root of the development of SARDS, resembling cancer-associated retinopathy in humans. Specifically, neuron-specific enolase autoantibodies were detected in 25% of the dogs with SARDS and not in the control animals, although it is unclear whether these were a cause or a result of outer retinal pathology (16). Other evidence suggesting an immune-mediated etiology for SARDS is relatively outdated (5,20) or non-peer-reviewed (21). More recent studies have been unsuccessful in demonstrating identifiable anti-retinal autoantibodies in the serum of dogs affected by SARDS compared with normal

dogs (12,14). Despite a lack of convincing evidence to support an immune-mediated process, intravenous immunoglobulin has been administered as therapy for dogs with SARDS and is purported to restore some degree of vision (21,22).

Limitations of the present study include inconsistently performed diagnostic workups for the majority of cases as well as inherent limitations that accompany retrospective studies. However, our report exceeds the case numbers of most retrospective studies published to date and is also the first retrospective report on SARDS in Canada. The data presented herein are useful as a descriptive baseline for future studies but, in the absence of control animals, must be interpreted carefully and within the general ophthalmology referral population.

In conclusion, dogs diagnosed with SARDS in western Canada had a similar clinical picture to previously reported cases of SARDS and corroborates the preponderance of middle-aged, small breeds that present with signs of polyuria, polydipsia, and have subtle clinicopathologic abnormalities. Chromatic pupillary light reflex testing may be more valuable than non-chromatic pupillary light testing in detecting pupil response abnormalities in dogs with SARDS; however, electroretinography is indispensable for diagnosing SARDS. Future directions for research include advanced diagnostic imaging such as optical coherence tomography and genetic studies, given the preponderance of SARDS in certain breeds. CVJ

References

1. Komáromy AM, Abrams KL, Heckenlively JR, et al. Sudden acquired retinal degeneration syndrome (SARDS) — A review and proposed strategies toward a better understanding of pathogenesis, early diagnosis, and therapy. Vet Ophthalmol 2016;19:319–331.
2. Montgomery KW, van der Woerdt A, Cottrill NB. Acute blindness in dogs: Sudden acquired retinal degeneration syndrome versus neurological disease (140 cases, 2000–2006). Vet Ophthalmol 2008;11:314–320.
3. Stuckey JA, Pearce JW, Giuliano EA, et al. Long-term outcome of sudden acquired retinal degeneration syndrome in dogs. J Am Vet Med Assoc 2013;243:1425–1431.
4. Heller AR, van der Woerdt A, Gaarder JE, et al. Sudden acquired retinal degeneration in dogs: Breed distribution of 495 canines. Vet Ophthalmol 2016;20:103–106.
5. Miller PE, Galbreath EJ, Kehren JC, Steinberg H, Dubielzig RR. Photoreceptor cell death by apoptosis in dogs with sudden acquired retinal degeneration syndrome. Am J Vet Res 1998;59:149–152.
6. O'Toole D, Roberts S, Nunamaker C. Sudden acquired retinal degeneration ('silent retina syndrome') in two dogs. Vet Rec 1992;130:157–161.
7. Acland G, Irby N, Aguirre G, Gross SL, Nitroy SF, Notafrancesco KL. Sudden acquired retinal degeneration in the dog: Clinical and morphologic characterization of the "silent retina" syndrome. Trans Am Coll Vet Ophthalmologists 1984;15:86–104.
8. Carter RT, Oliver JW, Stepien RL, Bentley E. Elevations in sex hormones in dogs with sudden acquired retinal degeneration syndrome (SARDS). J Am Anim Hosp Assoc 2009;45:207–214.
9. Van Der Woerdt A, Nasisse M, Davidson M. Sudden acquired retinal degeneration in the dog: Clinical and laboratory findings in 36 cases. Progress in Vet Comp Ophthalmol 1991;1:11–18.
10. Cabrera Blatter MF, del Prado A, Gallelli MF, et al. Blindness in dogs with pituitary dependent hyperadrenocorticism: Relationship with glucose, cortisol and triglyceride concentration and with ophthalmic blood flow. Res Vet Sci 2012;92:387–392.
11. Mattson A, Roberts SM, Isherwood JME. Clinical features suggesting hyperadrenocorticism associated with sudden acquired retinal degeneration syndrome in a dog. J Am Anim Hosp Assoc 1992;28:199–202.
12. Keller RL, Kania SA, Hendrix DV, Ward DA, Abrams K. Evaluation of canine serum for the presence of antiretinal autoantibodies in sudden acquired retinal degeneration syndrome. Vet Ophthalmol 2006; 9:195–200.
13. Terakado K, Yogo T, Nezu Y, Harada Y, Hara Y, Tagawa M. Efficacy of the use of a colorimetric pupil light reflex device in the diagnosis of fundus disease or optic pathway disease in dogs. J Vet Med Sci 2013;75:1491–1495.
14. Gilmour MA, Cardenas MR, Blaik MA, Bahr RJ, McGinnis JF. Evaluation of a comparative pathogenesis between cancer-associated retinopathy in humans and sudden acquired retinal degeneration syndrome in dogs via diagnostic imaging and western blot analysis. Am J Vet Res 2006;67:877–881.
15. Grozdanic SD, Matic M, Sakaguchi DS, Kardon RH. Evaluation of retinal status using chromatic pupil light reflex activity in healthy and diseased canine eyes. Invest Ophthalmol Visual Sci 2007;48:5178–5183.
16. Braus BK, Hauck SM, Amann B, et al. Neuron-specific enolase antibodies in patients with sudden acquired retinal degeneration syndrome. Vet Immunol Immunopathol 2008;124:177–183.
17. Söder J, Wernersson S, Hagman R, Karlsson I, Malmlöf K, Höglund K. Metabolic and hormonal response to a feed-challenge test in lean and overweight dogs. J Vet Intern Med 2016;30:574–582.
18. Vicennati V, Garelli S, Rinaldi E, Di Dalmazi G, Pagotto U, Pasquali R. Cross-talk between adipose tissue and the HPA axis in obesity and overt hypercortisolemic states. Horm Mol Biol Clin Investig 2014;17:63–77.
19. Pasquali R, Vicennati V, Cacciari M, Pagotto U. The hypothalamic-pituitary-adrenal axis activity in obesity and the metabolic syndrome. Ann New York Acad Sci 2006;1083:111–128.
20. Bellhorn RW, Murphy CJ, Thirkill CE. Anti-retinal immunoglobulins in canine ocular diseases. Semin Vet Med Surg (Small Anim) 1988; 3:28–32.
21. Grozdanic SD, Harper MM, Kecova H. Antibody-mediated retinopathies in canine patients: Mechanism, diagnosis, and treatment modalities. Vet Clin North Am Small Anim Pract 2008;38:361–387, vii.
22. Spurlock NK, Prittie JE. A review of current indications, adverse effects, and administration recommendations for intravenous immunoglobulin. J Vet Emerg Crit Care 2011;21:471–483.